

Appendix B: Exposure to Chemicals During the Gulf War

Chemical Warfare Agents

At the time of the Gulf War, the Iraqi forces had an experimental chemical weapons program and also had chemical munitions available for use in the field. United Nations Special Commission investigations indicated that chemical agents in the Iraqi chemical weapons program included sulfur mustard (a blistering agent), sarin, and VX (IOM, 1996b). Iraq was also assumed to have available the nerve agents, cyclosarin, soman, and tabun (CIA, 1997). The Iraqi's possession of munitions with nerve agents was well known. For example, in 1992, a U.K. newspaper, the Independent, reported that a United Nations demolition team announced they would destroy 400 sarin-filled, 122 mm rockets that were located at a large Iraqi weapons storage bunker, at Khamisiyah 25 miles north of Basra (Independent, 1992). It was reported that the bunker was damaged due to earlier Allied bombing raids and that it was necessary to blow up the rockets at the site because they were leaking.

In 1994, the U.S. Senate Committee on Banking, Housing, and Urban Affairs issued a report expressing the belief that there was “reliable evidence that U.S. forces were exposed to chemical and possibly biological agents” (U.S. Senate, 1994a). However, the 1994 National Institute of Health Technology Assessment Workshop report indicated that evidence of exposure to chemical warfare agents was controversial and drew no conclusions (NIH, 1994a,b). The Defense Science Board concluded that there was “no scientific or medical evidence” to indicate that U.S. troops in Kuwait or Saudi Arabia were exposed to chemical or biological warfare agents (DSB, 1994). In 1996, the Institute of Medicine (IOM, 1996b) indicated that there was no credible evidence that chemical weapons were used by Iraq in the Gulf War, noted that serious concerns persisted among veterans and some investigators that significant exposure to chemical agents may have occurred in non-combat situations, and pointed out that their committee had not had the opportunity to review evidence that troops may have been exposed to chemical agents during destruction of an Iraqi munitions bunker in March, 1991.

In response to inquiries from the Presidential Advisory Committee on Gulf War Veterans' Illnesses, the DoD announced in June 1996 that: chemical warfare agents had been known to be present at Iraqi weapons-storage sites at Khamisiyah, Iraq; that some of these sites were demolished by U.S. troops in March, 1991; and that troops in the vicinity may have experienced low-level exposure to chemical warfare nerve agents during the demolition events (PAC, 1996b; CIA, 1997; DoD, 1997a). The CIA (1997) acknowledged, based on information from United Nations Special Commission inspectors, that chemical warfare agents (sarin and cyclosarin) were likely present in at least two U.S. demolition events at Iraqi ammunition storage sites in the Khamisiyah area: one (Bunker 73) on March 4, 1991 and the other (“the pit”) on March 10, 1991 (DoD, 1997a). DoD (1997a) noted that there was evidence that another demolition event occurred at “the pit” on March 12, 1991. In addition, chemical weapons storage sites at Muhammadiyat, and Al Muthanna, Iraq (northwest of Baghdad) were destroyed by Allied

bombing at the beginning of the Gulf War creating a potential risk of exposure for troops located 400-500 km south of the sites (RWG, 1997).

Because air monitoring data are not available for these events, models were developed, based on the limited amount of data available concerning the amount of nerve agent that may have existed at the Khamisiyah sites, to calculate estimates of ground level concentrations of nerve agents (sarin/cyclosarin) as a function of distance and direction away from detonation sites for the March 4 and 10, 1991 events (PAC, 1996b; CIA and DoD, 1997). Modeling results, as of October, 1996, indicated that exposure levels in the explosion plume within 25 kilometers of the demolition site may have been sufficient to cause runny nose, tightness in the chest and dimness of vision (CIA and DoD, 1997), but the U.S. Army Medical Corps reported to the PAC (1996b) that signs and symptoms characteristic of exposure to nerve agents such as sarin and soman were not seen by medical personnel during the Gulf War (PAC, 1996b), and no reports of distinct acute neurological poisonings during the March 1991 Khamisiyah demolitions were located. Efforts to decrease the uncertainty in the modeling efforts are ongoing (CIA and DoD, 1997). The PAC (1996b) concluded that evidence of chemical warfare agent release at Khamisiyah “is overwhelming” and that “low-level exposure to troops within a 50-km radius should be presumed while efforts to develop more precise measures of exposure and more detailed knowledge of the demolition activities continue.”

Beginning in August 1996, DoD notified approximately 20,000 individuals (those expected to have been within a 50-km radius of the Khamisiyah demolition sites) that they could have been exposed to low-levels of chemical warfare agents (DoD, 1997a; PAC, 1997). Surveys were mailed to these individuals concerning health symptoms that they recollected experiencing; the CIA and DoD (1997) reported that 7,400 responses were received and that 99 percent of responses indicated “no physical effects that could be correlated with exposure to sarin”. The PAC (1997) recommended that DoD should contact all individuals within a 300-mile radius of the Khamisiyah “pit”, notifying them if they are, or are not, expected to have been under the plume of the Khamisiyah demolition events.

The DoD has publicly released case narratives of investigations of numerous events of possible chemical warfare agent exposures during the Gulf War period. These include:

- C Czech and French detections of nerve gas and blister agents in January 1991 in the vicinities of Hafar al Batin and King Khalid Military City (DoD, 1998a),
- C chemical-agent detections by a Fox vehicle in an ammunition supply point in an orchard southwest of Kuwait City in February 1991 (DoD, 1997b),
- C reports of loud-noise, SCUD missile impacts, and “noxious” cloud events in the Al Jubayl, Saudi Arabia region on several dates between January and March, 1991 (DoD, 1997c),
- C several instances of suspected chemical warfare use during combat to retake Kuwaiti Air Base in Al Jaber in February, 1991 (DoD, 1997d),
- C multiple (18) chemical-alarm alerts reported by the 11th Marines over a 42-day period between January 17 and February 27, 1991 (DoD, 1998b),

- C reports of chemical weapon (mustard) storage at the Iraqi An Naisiriyah Southwest ammunition storage point, a site at which U.S. carried out demolition operations in March and April 1991 (DoD, 1998c), and
- C development of skin burns on a U.S. Army Sergeant after performing reconnaissance in an Iraqi bunker in March, 1991 (DoD, 1997e).

DoD investigators reached conclusions (noted as interim) regarding the possibility of exposure during these events that ranged from “definitely not” (Al Jubayl events; Al Jaber Air Base) to “likely” (Army Sergeant with mustard agent burns). Several events were assigned an “unlikely” assessment (11th Marine events; An Naisiriyah ammunition storage point; Fox detections in orchard). No narrative assigned the “definite” category of exposure assessment. The PAC (1997) recommended that an entity other than the DoD should provide oversight of investigations about possible chemical warfare agent exposures. The Presidential Special Oversight Board has been established to accomplish such a role (PSOB, 1998).

The PAC (1996a, 1997) noted that chemical-warfare-agent detectors used by the U.S. during the Gulf War period (e.g., M8A1 chemical agent alarms, Fox vehicles with MM-1 mass spectrometers, and other detectors) could detect nerve gas agents only at concentrations that would cause acute lethal or near-lethal poisonings and not at low levels that might have subclinical health significance. The principal battlefield detector (M8A1) could not detect mustard agent and was so non-specific in its detection that it was often ignored during the war. The PAC (1997) recommended that DoD support the development of new detectors for “low-level, subclinical exposures” to chemical warfare agents.

After review of information, data, and modeling calculations available for Khamisiyah and other sites, as well as DoD’s case narratives and information papers on the potential exposure of troops to chemical agents, the Senate’s Special Investigation Unit concluded that there was insufficient evidence to prove or disprove that there was an actual low-level exposure of any troops to chemical weapon nerve agents or that any of the health effects some veterans are experiencing were caused by such exposure (U.S. Senate, 1998).

Pyridostigmine Bromide

Pyridostigmine is an anti-nerve agent (a carbamate molecule) that binds reversibly at sites of the important nerve enzymes (cholinesterases) that are irreversibly bound by organophosphate nerve agents such as sarin. At suitable dosage levels, the binding of carbamates or organophosphates to cholinesterases causes an overstimulation of cholinergic nerves in the peripheral and central nervous systems. Pyridostigmine is expected to provide protection against severe acute organophosphate poisoning when given before exposure to organophosphate agents, based on results from animal experiments showing that pyridostigmine pretreatment coupled with post-exposure treatment with atropine and pralidoxime chloride increased survival after exposure to lethal concentrations of the nerve agent, soman (Harris et al., 1984; Dirnhuber et al., 1979). The reversible binding of pyridostigmine is thought to temporarily protect the enzymes from

permanent damage that can be caused by irreversibly binding organophosphate nerve agents (Glikson et al., 1991; Taylor, 1996).

Prior to the Gulf War, the U.S. Food and Drug Administration (FDA) had approved the use of this drug for the treatment of myasthenia gravis, an autoimmune disease characterized by muscle weakness, but had not approved its repeated use as a pretreatment, protective therapy against organophosphate nerve agents in healthy subjects (U.S. Senate, 1994b). FDA regulations require obtaining an informed consent agreement from any individual who might use such an “investigational new drug”. In 1990, the DoD requested that FDA waive its informed consent requirement for pyridostigmine, and, in January 1991, the FDA Commissioner agreed to waive informed consent due to the lack of an alternative satisfactory therapy against organophosphate nerve agents and the infeasibility of obtaining informed consent agreements under combat conditions (Annas, 1992; U.S. Senate, 1994b).

Although results from animal studies indicate that pretreatment with pyridostigmine is effective at decreasing lethality from certain organophosphate nerve agents (Dirnhuber et al., 1979; Harris et al., 1984), excessive doses of pyridostigmine are expected to cause some of the same acute toxic effects that are produced by organophosphate nerve agents due to stimulation of peripheral cholinergic nerves (Taylor, 1996). Studies with rhesus monkeys, however, showed that exposure to pyridostigmine at exposure levels that produced 70-80% inhibition of blood cholinesterase did not significantly affect performance in neurobehavioral tests, whereas exposure to the organophosphate agent, soman, at levels that produced similar blood cholinesterase inhibition, produced severe behavioral toxicity (Blick et al., 1994). These results suggest that the potency of pyridostigmine to affect the central nervous system is much less than the potency of organophosphate nerve agents. Recent results from rodent studies indicate that pyridostigmine pretreatment may not be equally effective at protecting against the lethality of all organophosphate nerve agents. Koplovitz et al. (1992) reported that pretreatment of mice or guinea pigs with pyridostigmine increased the efficacy of treatment with atropine and pralidoxime chloride after exposure to the organophosphate nerve agent, tabun, but with exposure to other organophosphate agents (sarin and VX), the efficacy of atropine and pralidoxime chloride treatment was decreased by pyridostigmine pretreatment.

DoD reported that all U.S. troops were supplied with pyridostigmine bromide pills, and that approximately 250,000 personnel took at least some pyridostigmine during the Gulf War (PAC, 1996b). During the Gulf War, pyridostigmine was to be used at the commanding officer’s judgement and was to be self-administered by individuals in 30-mg doses three times daily (U.S. Senate, 1998). At the recommended dosage levels, acute, transient “side effects” from pyridostigmine appear to be mild in most individuals who report experiencing them. Reports from U.S. medical personnel providing care to 41,650 U.S. soldiers who took the recommended dosage for 1 to 7 days in January 1991 indicated that about 50% experienced gastrointestinal symptoms, 5-30% experienced urinary urgency and frequency, <5% experienced headaches, runny nose or tingling of the extremities, 1% (483 soldiers) required clinical visitation, and <1% (28 soldiers) had to discontinue use due to severe acute reactions (Keeler et al., 1991).

There is evidence that stress may enhance the acute adverse effects from pyridostigmine treatment. Symptoms of central nervous system dysfunction (e.g., headaches, insomnia, drowsiness, nervousness, difficulties in focusing attention) were reported by about 24% of 213 soldiers who took pyridostigmine under wartime conditions and were surveyed within 24 hours, whereas in a double-blind, placebo-controlled study under non-stressed conditions, about 8% of subjects given the same dose of pyridostigmine bromide reported similar symptoms (Friedman et al., 1996). Friedman et al. (1996) hypothesized that stress may disrupt the blood-brain barrier in some manner, allowing greater quantities of pyridostigmine to enter the brain compared with quantities that enter under non-stress conditions.

At dosage levels used for organophosphate nerve agent protection, limited testing has suggested that the short-term use of pyridostigmine may not have delayed or chronic neurological effects. As noted above, pyridostigmine has been used widely for decades in the treatment of the autoimmune disease, myasthenia gravis. The muscle weakness and fatigue associated with this disease is due to an autoimmune reaction with the acetyl choline receptor in neuromuscular nerve junctions (Drachman, 1994; Taylor, 1996). In these diseased subjects, the ability of pyridostigmine to reversibly inhibit acetylcholinesterases is thought to sufficiently increase endogenous concentrations of acetyl choline so that the abnormally low numbers of functional acetyl choline receptors are stimulated and muscle function is improved. No reports were found of chronic neurological or psychological effects in myasthenia gravis patients chronically treated with pyridostigmine bromide. Animal studies have reported changes in structure, ultrastructure and electrophysiological properties of neuromuscular synapses after repeated exposures to carbamates similar in structure and activity to pyridostigmine (Engel et al., 1973; Hudson et al., 1978; Tiedt et al., 1978), but a double-blind, placebo-controlled study found no evidence for adverse effects in extensive tests of neuromuscular function in 35 healthy human volunteers who took 30 mg pyridostigmine bromide, three times a day for up to 8 days (Glikson et al., 1991). In a study of 4 human volunteers who took 30 mg pyridostigmine bromide every 8 hours for 3 days, Borland (1985) reported that no drug-induced changes in electrical activity of the brain were detected and that acute reversible changes were noted in tests of visual motor coordination. The motor coordination changes were noted as minimal.

Biological Warfare Agents

At the time of the Gulf War, the Iraqi forces had experimental biological weapons programs and also had biological munitions available for use in the field. United Nations Special Commission investigations indicated that biological agents in the Iraqi biological weapons program included botulinum toxin, anthrax, aflatoxin, ricin, mycotoxins, hemorrhagic conjunctivitis virus, rotavirus, and wheat cover smut (IOM, 1996b). During the Gulf War, biological warfare agent field detectors were relatively primitive and could not be relied upon to accurately detect exposure in a timely fashion. U.S. Army hospital admission records identified one admission for anthrax, a disease indigenous to the Gulf region (PAC, 1996b; U.S. Senate, 1998).

Recent review panels (U.S. Senate, 1998; PAC, 1996b, 1997) have concluded that biological warfare agents were not likely used during the Gulf War because: there is no evidence to date from intelligence agencies that indicates their use; there were no verified detections of anthrax or botulinum toxin during the war; and examination of Iraqi soil samples and enzyme assays by U.S. laboratories did not find evidence of the presence of biological warfare agents.

As discussed previously, the Presidential Advisory Committee further recommended that, “To ensure credibility and thoroughness, further investigation of possible chemical or biological warfare agent exposures during the Gulf War should be conducted by a group independent of DoD.” (PAC, 1996b, 1997). The Presidential Special Oversight Board has been established to accomplish such a role (PSOB, 1998).

Infectious Diseases

Many infectious diseases are prevalent in southwest Asia including, but not limited to, agents that cause diarrhea, leishmaniasis, sandfly fever, and malaria. DoD medical personnel monitored troops for the preceding diseases as well as for dengue fever, Sindbis, West Nile fever, Rift Valley fever, and Congo-Crimean hemorrhagic fever, and took measures to prevent illness from endemic diseases (Hyams et al., 1995; PAC, 1996b).

During the Gulf War, infectious diseases were not a significant problem; diarrhea was the most commonly reported condition. Occurrence of diarrhea was 4% per week early in the deployment and declined to <0.5% per week after controls on food sources were imposed (Hyams et al., 1995). Although sand fly fever had been a concern, no cases were found during the war (Hyams et al., 1995). Seven individuals with malaria were diagnosed, one individual had West Nile fever, and one death occurred from meningococcal meningitis (Hyams et al., 1995).

A small number of cases of leishmaniasis (a chronic disease transmitted, like sand fly fever, by the bite of the sand fly) has been diagnosed among U.S. Gulf War veterans: 12 cases of viscerotropic leishmaniasis and 19 cases of cutaneous leishmaniasis (PGVCB, 1995). Most of these cases have displayed objective signs of the chronic disease: elevated temperatures, lymphadenopathy, and hepatosplenomegaly (Magill et al., 1993). The PAC (1996b) arrived at the conclusion that it is unlikely that infectious diseases endemic to the Gulf are responsible for long-term health effects most frequently reported by Gulf War veterans.

Infections by mycoplasma species, microsporidia, and streptococcal bacteria have been hypothesized as possible explanations for illnesses noted in some Gulf War veterans. Nicolson and Nicolson (1996) reported that mycoplasma gene sequences were detected in blood leukocytes from 14 subjects in a group of 30 Gulf War veterans with chronic symptoms similar to those associated with chronic fatigue syndrome and that 11/14 of these subjects recovered after multiple treatment cycles of antibiotics (doxycycline or ciprofloxacin). Nicolson et al. (1998) also reported that mycoplasma gene sequences were detected in blood leukocytes of 76 subjects in a group of 170 subjects comprised of Gulf War veterans with chronic-fatigue-syndrome-like symptoms and

their immediate family members. Among 73 mycoplasma-positive subjects who received two to six 6-week cycles of antibiotic therapy (doxycycline, ciprofloxacin or azithromycin), 58 were reported to have recovered. Hyman (1996) reported the detection of streptococcal bacteria remnants in urine of about ten Gulf War veterans who had chronic-fatigue-syndrome/fibromyalgia-like symptoms (and their immediate family members); treatment with antibiotics was reported to improve the health of the subjects initially, but most relapsed. An initial DVA report of finding microsporidia in stool specimens of some Gulf War veterans was not confirmed with subsequent examinations of stool and gastrointestinal biopsy material (PAC, 1996b) or in CDC examinations of stool specimens from Gulf War veterans in Air Force units from Pennsylvania and Florida (Fukuda et al., 1998). In 1996, the PAC (1996b) expressed the belief that it was unlikely that these three infectious agents “are responsible for widespread disease among Gulf war veterans or their families.”

Immunizations

Seven vaccines (polio, diphtheria-tetanus, adenovirus 4 and 7, meningococcus A, CYW135, influenza, and measles-rubella) are administered to U.S. Army recruits during basic training, and others are administered upon deployment to high risk areas (hepatitis A and B, yellow fever, Japanese encephalitis, plague, rabies, and cholera) (IOM, 1996b). DoD reported to the PAC (1996b) that approximately 150,000 Gulf deployed personnel received at least one anthrax vaccination and about 8,000 personnel received at least one dose of botulinum toxoid vaccine, but adequate records to document which troops received the anthrax and botulinum toxoid vaccines were not available.

The anthrax vaccine, licensed by FDA since 1970, produces injection site reactions (e.g., swelling, tenderness) in about 6% of recipients (IOM, 1996b). The botulinum toxoid vaccine, which has been assigned an “investigational” status by the FDA and has been used as an investigational vaccine to protect high-risk laboratory workers, consists of five types of toxins (from *Clostridium botulinum*) that are converted to a “toxoid” status by reaction with formalin (IOM, 1996b). Annas (1992) has noted that the use of the vaccine in laboratory workers was discontinued in the mid-1970s before sufficient data on safety and efficacy had been collected for licensing purposes. The experience of the U.S. Army Medical Research Institute of Infectious Diseases with the botulinum toxoid vaccine indicates that transient reactions include pain, redness, and swelling at the injection site in about 10% of recipients, and headache, myalgia, fever, and malaise in about 3% (IOM, 1996b). Given the possibility that Iraq might use botulinum toxin as a biological weapon, the DoD had requested, in 1990, that FDA waive informed consent requirements for the use of a botulinum toxoid vaccine; this request was granted by the FDA in 1991 noting that obtaining informed consent agreements was not feasible under combat conditions (U.S. Senate, 1994b). Annas (1992) reported that the DoD sent a letter to the FDA noting that, during the Gulf War, the military command decided to administer the botulinum toxoid vaccine on a voluntary basis.

Depleted Uranium (DU)

DU, a byproduct of uranium refinement, is a very dense material that is used to increase the penetration capability of antitank munitions and as a protective shield on tanks against enemy fire (DoD, 1998d). The major toxicity of acute exposure to DU is from its chemical properties, rather than its radioactive properties, but there is uncertainty regarding toxicity from long-term exposure (IOM, 1996b). DU, which has about half the radioactivity of natural uranium, was first used in combat during the Gulf War, during which U.S. troops fired approximately 285 tons of DU munitions. Many U.S. troops handled munitions containing DU, but significant exposure with handling is not expected since the DU is encased in a protective shell (IOM, 1996b). Radiation exposure from intact DU munitions and armor is minimal and within accepted standards of health safety (GAO, 1993; IOM, 1996b).

During the Gulf War, friendly fire incidents wounded 35 U.S. soldiers of whom 22 were suspected to have retained DU fragments. Thirty-three of these wounded soldiers are undergoing a DVA-sponsored medical surveillance program at the Baltimore VA Medical Center. After 3 years, 15 of the 33 soldiers had detectable shrapnel. To date, the follow-up studies have found no evidence for neurological, renal, genotoxic, or immunological effects, but uranium excretion has been noted to be elevated in those known to have retained shrapnel (Keogh, 1995; Joseph et al., 1998). A report of the findings of this surveillance program is in preparation and will likely be available in 1999 (DVA, 1999).

The PAC (1996b) and the GAO (1993) noted that DoD had appropriate procedures for protecting personnel who worked with DU contaminated vehicles during the Gulf War but, apparently, few U.S. service personnel were adequately trained in these procedures. Activities of the 144th Service and Supply Company in fighting fires, recovering vehicles, and cleaning 29 tanks damaged by DU munitions may have led to DU exposure of 27 soldiers. Results of testing 12 of these soldiers were negative and the remaining 15 chose not to be tested (IOM, 1996b). Another two dozen soldiers from the 24th Infantry Division have reported that they were unknowingly exposed to DU-contaminated debris in the course of vehicle recovery and maintenance operations (PAC, 1996b). Additionally, troops may have inhaled particles containing DU while working near a fire at the Doha-Kuwait armored vehicle depot, or while climbing onto allied or enemy vehicles that had been hit by munitions containing DU (U.S. Senate, 1998).

DoD (1998d) classified possible DU exposures during the Gulf War into three levels:

- C Level I represents immediate and direct exposures of soldiers in or near combat vehicles at the times these vehicles were struck by DU penetrators or who entered vehicles immediately after they were struck by DU munitions. These soldiers could have been struck by DU fragments, inhaled DU aerosols, ingested DU residues, or had DU particles land on open wounds, burns, or other breaks in their skin.
- C Level II represents a lower level of exposure for soldiers and a small number of DoD civilian employees who worked in and around wrecked vehicles containing DU fragments and particles. These people may have inhaled DU residues resuspended during their

activities, transferred DU from hand to mouth, or spread contamination on their clothing. This Level includes soldiers who were involved in cleaning up DU residues that remained after a motor pool fire in which DU munitions detonated and burned.

- C Level III represents people who received short-term and very low exposures and included individuals who entered DU-containing Iraqi equipment, troops downwind from burning Iraqi or U.S. equipment struck by DU rounds, or personnel downwind from burning DU ammunition.

DoD (1998d) identified thirteen exposure events during the Gulf War period - two classified as Level I, seven as Level II, and four as Level III. Health risk assessments are being prepared for all thirteen events. The risk assessments will describe the activities of the participants, specify the sources of potential DU exposure, and estimate the dose from inhalation, ingestion, and wound contamination as appropriate for each exposure (DoD, 1998d).

In 1998, the DoD and DVA expanded a medical follow-up program conducted by the Baltimore VA Medical Center to evaluate the remaining veterans who received the largest DU exposures during the Gulf War, those involved in Level I and II exposure events. The evaluations will include a medical examination, determination of uranium levels in the urine, and completion of a detailed DU exposure questionnaire (Rostker, 1998; DVA, 1998b).

Pesticides

The DoD reported that pesticides shipped to the Gulf region for use during the war included 45,770 pounds of malathion, 8,410 pounds of chlorpyrifos, 1,858 pounds of D-phenothrin, 903 pounds of methomyl, and 539 pounds of lindane (IOM, 1996b). Pyrethrin, dichlorovos (DDVP), carbaryl, propoxur, and diazinon were also available but in amounts less than 330 pounds each (IOM, 1996b). All pesticides shipped were approved by EPA or FDA for general use in the United States at the time of the war (PAC, 1996b). It is not known how much of this inventory of pesticides was actually used or what troop exposures may have resulted (IOM, 1996b).

The use of pesticides in the Gulf was reported to have followed strict guidelines. They were used only after arthropod surveys that identified individual pests and estimated arthropod prevalence. Distribution of pesticides was prohibited unless approved by the local commander. Distribution or use for other than personal purposes was restricted to trained or certified personnel or contractors (IOM, 1996b).

DoD reported that about 2.2 spray-cans of permethrin and 2 tubes of DEET (33%) for each U.S. service member were shipped to the Gulf (PAC, 1996b). Some troops were reported to have both applied the insect repellents DEET on their skin and permethrin on their clothing between August and October, 1990, the peak occurrence of arthropods (IOM, 1996b). In addition, some service personnel chose to wear animal flea collars for protection from insects, although DoD discouraged this practice (U.S. Senate, 1998).

Smoke from Oil Well Fires

Near the end of the Gulf War in February, 1991, the Iraqi troops set more than 1,000 Kuwaiti oil wells and refineries on fire (Spektor, 1998). The burning wells were located in eastern Kuwait, with the majority to the south of Kuwait City. Smoke plumes rose and combined in a “superplume” that could be seen for hundreds of kilometers and sometimes even partially blocked out the sun (U.S. Senate, 1998).

Systematic environmental monitoring did not begin until May 1991, so limited exposure data are available for the period when most U.S. troops were in the Gulf area (Spektor, 1998, USAEHA, 1994). The U.S. Army’s Environmental Hygiene Agency (USAEHA) carried out the largest monitoring effort, collecting nearly 4,000 ambient air and soil samples between May and December, 1991 (USAEHA, 1994).

Air monitoring data from the USAEHA and other U.S. and international agencies indicated that air levels of nitrogen oxides, carbon monoxide, sulfur dioxide, hydrogen sulfide, other pollutant gases, and polycyclic aromatic hydrocarbons (PAHs) were lower than anticipated and did not exceed levels seen in urban air in a typical U.S. industrial city (USAEHA, 1994; Spektor, 1998). A health risk assessment conducted by the USAEHA (1994) based on the air monitoring data for volatile organic compounds, particulate heavy metals, and PAHs predicted an excess risk for cancer of three cases per million persons exposed. Risks for non-cancer health effects were estimated by a hazard index approach comparing estimated exposure levels during the fires to U.S. EPA reference exposure levels expected to be without adverse health effects (an index greater than 1 indicates increased risk for general populations including sensitive individuals): hazard indices ranged from 0.6 to 2.0 in Saudi Arabia and 2.0 to 5.0 in Kuwait (USAEHA, 1994).

Inhalation of volatile organic chemicals, particularly benzene, contributed to over 99 percent of the non-cancer health risk at all monitoring sites. The USAEHA (1994) noted that the EPA reference exposure levels each have at least 10-fold margins of safety incorporated in their derivation and that hazard indices in the range of 1 to 10 should not present “an unreasonable health risk, particularly for short-term exposures”, noting that DoD personnel were exposed to the smoke for a minimum of about a month to a maximum of about 9 months.

Etzel and Ashley (1994) found elevated concentrations of several volatile organic compounds (VOCs) in blood samples collected from 40 American firefighters working in the Kuwait oilfields in October, 1991 compared with blood levels in a random sample of 114 U.S. residents. The measured VOCs (benzene, toluene, xylene, and styrene) are components of smoke from oil well fires; blood levels in firefighters were about two times average levels in the reference group. Concentrations of these VOCs were not elevated, however, in blood collected in May 1991 from 14 U.S. personnel who worked in Kuwait City compared with reference levels.

Analyses of biologic samples from deployed troops, local inhabitants, and autopsy cases have not indicated a risk for health effects from atmospheric pollution caused by the fires (Coombe and Drysdale, 1993; Mullick, 1996). No cases of illness with symptoms resembling the most prevalent

symptoms reported by U.S. Gulf War veterans in the DoD and DVA health registries have been found in a group of 110 oil-well firefighters who worked daily at Kuwaiti wells in 1991 for 28-day periods without breathing-protection equipment or in other oil-well firefighters with years of experience (Friedman, 1994, 1996).

One study reported an increase in frequency of sister-chromatid exchange in blood cells collected from soldiers who were deployed from Germany to the Persian Gulf to participate in monitoring of the Kuwait oil-well fires between June and September, 1991 (after combat had ceased), but the cause of this apparent increase could not be determined (McDiarmid et al., 1995). Sister chromatid exchanges have been used as an indicator of exposure to a number of environmental mutagenic agents, including polycyclic aromatic hydrocarbons (PAHs). PAHs and other mutagenic agents are present in smoke from oil-well fires and from other fires as well. A further study of a subset of these soldiers measured levels of three biomarkers for exposure to PAHs (two measures of PAH-DNA adducts in blood cells and urinary levels of 1-hydroxypyrene-glucuronide, a metabolite of PAHs) before deployment to the Gulf, during deployment in Kuwait (after 8 weeks of duty), and 4 weeks after returning to Germany (Poirer et al., 1998). Levels of PAH biomarkers were lowest during deployment in Kuwait, suggesting that this group of soldiers were not exposed to elevated levels of PAHs while deployed in Kuwait.

Petroleum Products and Other Chemicals

The fuel used most widely during the Gulf War for both vehicles and equipment was Jet A-1, a kerosene-based aviation fuel. Of the 1.8 billion gallons of fuel used during the Gulf War, roughly 75 percent was jet fuel (mostly Jet A-1), 24 percent was diesel fuel, and 1 percent was gasoline. The gasoline was commercial leaded gasoline (PAC, 1996b). About 145,000 gallons of gasoline were used per day for eight months starting in August, 1990 (IOM, 1996b). Besides use in vehicles and machine engines, petroleum products were used to burn human waste and trash and as a fuel in stoves (U.S. Senate, 1998). Diesel fuel was used in large amounts to suppress dust, with one reported case involving 30,000 gallons used on roads daily. Troops living in tents near the roads, and particularly truck drivers who carried out the spraying, complained of nausea from breathing the resulting fumes (PAC, 1996b).

When fuels were used for heaters, cooking stoves, and portable generators, the fumes and exhaust produced by these fuels, particularly when used in unventilated tents, would have exposed some service members to benzene, toluene, xylene, ethyl benzene, and combustion products including carbon monoxide, sulfur dioxide, nitrogen dioxide, particulates, lead, and other pollutants (PAC, 1996b; U.S. Senate, 1998; IOM, 1996b). Air and limited blood monitoring found no evidence of elevated exposure to volatile organic compounds (PAC, 1996b). A recent study simulated Gulf War exposures to aerosols from unvented heaters in tents and found elevated concentrations of particulate matter, nitrogen oxides and carbon monoxide (Cheng, 1998). Fuel type, heater type, and air exchange rate were important factors in determining air concentrations in the tent. Cheng (1998) noted that information from this study will be used to calculate respiratory doses that may

have been experienced by troops residing in heated tents during the Gulf War and to calculate estimates of health risks from this type of exposure.

Chemical Agent Resistant Coating (CARC) paint, which releases a compound (toluene diisocyanate) that can adversely affect the lungs, was applied to vehicles and equipment before shipment to the Gulf area or at a port in Dhahran (U.S. Senate, 1998). Accidental exposure to a chemical decontaminant agent containing propylene and ethylene glycols reportedly caused rashes in a group of soldiers (U.S. Senate, 1998).

The Desert Environment

In the initial months of the deployment, troops were exposed to summer daytime temperatures that reached as high as 130 degrees Fahrenheit. In August and September, the mean high temperatures were approximately 100 degrees Fahrenheit with very intense solar heat and low humidity. Preventive medicine efforts resulted in very few heat casualties (U.S. Senate, 1998; Joseph et al., 1998). In surveillance data on 40,000 Marines, less than three cases of heat injury requiring aid station treatment occurred weekly per 1,000 people (U.S. Senate, 1998). Sand flies were present, as evidenced by a few cases of leishmaniasis (IOM, 1996b).

High levels of airborne particulates were detected at several monitoring sites in the Gulf theater and sample analysis indicated that, frequently, the particulates were predominately sand (USAEHA, 1994). Korenyi-Both et al. (1992, 1997) theorized that acute respiratory problems experienced by U.S. troops in Al Eskan village between January and March, 1991 were caused as a result of immunosuppression from inhalation of airborne fine sand particulates along with organic pathogenic components; it was further theorized that the acute event may have induced a later-developing state of immunodeficiency that may be related to symptoms of ill health reported by Gulf War veterans. Studies to test this hypothesis were not located.

Psychological and Physical Stressors

The stresses of the Gulf War experience, some of which were unique, included sudden mobilization for military service in a hot, sandy, and foreign desert; exposures to the largest, most dramatic oil well and refinery fires in history, which spilled smoke and oil over a vast area; and potential exposure to chemical and biological warfare agents. Stresses reported by a group of over 2,000 Gulf War veterans as they returned home included nearly 300 events they considered stressful beyond the traditional combat experiences. The reported stress-related events were grouped into the following categories (U.S. Senate, 1998):

- C Combat and mission stressors such as actual threat to life from missiles (e.g., friendly fire incidents) or direct exposure to another's death or injury as part of a combat mission.
- C Non-combat war-zone stressors such as a unit member seriously injured or killed in a non-mission accident.

- C Domestic stressors such as divorce or long separation from or illness of family members and loved ones.
- C Anticipation of war/combat activities related to missile attack alerts or fear of attack by chemical or biological agents.
- C Physical attributes of the war zone such as severe climate or environmental conditions, long tours of duty, physical limitations and dangers from wearing chemical protective gear in a desert environment, or uncertainty about the war's duration.
- C Intra-unit stress related to personal conflicts in a unit, leadership failure or problems, or harassment.

Stress from personal and family concerns likely played a more prominent role in the Gulf War than in other wars, because it involved a greater number of married personnel and parents. In the Vietnam War, 16% of those deployed were married with children, whereas 60% of service members and reservists in the Gulf War were married with dependents, including approximately 32,000 single parents who had to make arrangements for their children during the deployment (U.S. Senate, 1998).

General Exposures of Military Service

In the military environment, personnel are required to perform multiple combat and non-combat activities that may involve potentially hazardous exposures, some of which may be similar to those in the civilian workplace. In addition, however, military personnel who participate in combat and combat support operations are exposed to inherent hazards that are associated with the operation of weapons systems and the battlefield environment. Common exposures to risk factors in the combat environment include propellants from ammunition; combustion products from vehicles; solvents; chemical warfare agents; noise, vibration, and non-ionizing radiation from communications and radar tracking equipment and laser target designators; blast impact, acoustical energy, airborne toxicants, extremes in barometric pressure, oxygen deficiency, and whole-body vibration from operation of tanks, aircraft, and submarines; biological hazards; extremes in temperature, humidity, and weather; and psychological stressors related to fear and isolation. Since a large proportion of the Gulf War veterans were members of the reserves or National Guard and also had non-military jobs, their civilian occupational exposures are potential confounders in the evaluation of their health problems (Joseph et al., 1998).

Hyams et al. (1996) noted that the clinical findings for Gulf War veterans are consistent with the experiences of U.S. veterans of previous wars. Reviewing U.S. clinical reports of war-related illnesses associated with the Civil War, World Wars I and II, the Korean Conflict, and the Vietnam War, Hyams recognized two general categories of war-related illnesses that were diagnosed after each of these wars:

1) psychological illnesses, given various names through the years from *nostalgia* in the Civil War, through *shell shock* in WWI, and *battle fatigue* in WWII and Korea, to *post-traumatic stress disorder* after the Vietnam and Gulf Wars; and

2) physiological illnesses, including *Da Costa* syndrome (irritable heart) after the Civil War, *Effort syndrome* during and after WWI and II, *Agent Orange exposure* after Vietnam, and *Gulf War syndrome*.

The physiological illnesses were primarily defined by self-reported, chronic symptoms including fatigue, shortness of breath, headache, sleep disturbances, impaired concentration, and forgetfulness. Hyams noted that these symptoms are non-specific and are frequently found in all adult populations, as well as among persons with illnesses associated with psychological stress, and that, in each of these wars, the onset of these illnesses was preceded by a high frequency of diarrhea. Hyams concluded that “poorly understood war syndromes” have recurred since the U.S. Civil War, that no single disease or underlying cause that is unrelated to psychological stress is apparent from reviewing the available clinical reports, and that the relationships between chronic, non-specific symptoms and physiological and psychological illness need to be better understood.

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